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The Case | Best not shaken or stirred! Chronic lymphocytic leukemia and hyperkalemia

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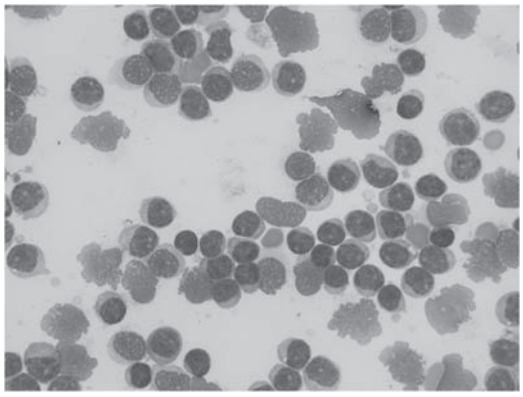

Figure 1 | Wright's stain of bone marrow aspirate revealing multiple smudge cells in the background of neoplastic lymphocytes.

Table 1 | Pertinent laboratory data

Test name	Laboratory value	Normal range
White blood cell count	136 K/μl	4–11
Hemoglobin	10.6 g/dl	13–17
Platelet count	19 K/μl	130–400
Uric acid	3.3 mg/dl	3.5–7.2
Lactate dehydrogenase	215 U/L	100–190
Creatinine kinase	11 U/L	35–2332
Creatinine	1.0 mg/dl	0.8–1.3
Bicarbonate	19 mEq/l	22–29

A 79 year old male with chronic lymphocytic leukemia (CLL) presented to the emergency department with sepsis. Five days prior to presentation he was treated with cyclophosphamide, vincristine, rituximab and prednisone for progressive CLL, as noted on a bone marrow biopsy performed the previous week. (Figure 1) He was admitted to the intensive care unit (ICU) and empirically treated with cefepime, ciprofloxacin and vancomycin. Blood cultures revealed *E. coli* sensitive to cefepime, and the antibiotics were altered appropriately. During his ICU stay his serum potassium ranged from 3.5–4.9 mmol/l. Upon transfer to the floor his serum potassium was noted to be 5.6 mmol/l at 1300. When rechecked at 1900 it was 6.5 mmol/l. The next morning at 0500 it was 6.4 mmol/l. Later that day, at 1215, it was 7.1 mmol/l. An electrocardiogram obtained did not demonstrate signs of hyperkalemia (peaked t-waves or sine waves).

His medications included allopurinol 200 mg, daily; cefepime 2 gm, q8 hours; flunisolide 2 sprays each nostril, BID; metoprolol 12.5 mg, q6 hours; omeprazole 20 mg, daily; prednisone 30 mg, daily; 0.9% sodium chloride 30 ml/h i.v.; PRN acetaminophen, albuterol nebulized, insulin, ipratropium nebulized, lorazempam i.v., metoprolol 5 mg i.v., and ondansetron 4 mg i.v.

Pertinent laboratory data are listed in Table 1.

What is the cause of the hyperkalemia?

SEE NEXT PAGE FOR ANSWERS

The Diagnosis | Pseudohyperkalemia due to blood sample agitation during pneumatic tube transport

Table 2 | Potassium levels and transport method

Day	Time (hours)	Specimen and transport method	Potassium (mmol/l)
1	1434	Serum vacutainer, tubed	5.8
1	1505	ABG, tubed	3.1
2	0600	ABG, tubed	3.2
2	0700	Serum vacutainer, tubed	7.6
2	0700	Serum vacutainer, walked	3.2

Abbreviation: ABG, artery blood gas.

In this case pseudohyperkalemia was suspected. To further establish the diagnosis, serum potassium measurements were obtained from a peripherally inserted central catheter through gentle suction with a 10 ml syringe and transferred into a heparinized lithium vacutainer; 31 min later a whole-blood potassium level was obtained from a radial artery blood gas (heparinized, no lithium). Both were transported by the pneumatic tube system. The discrepancy was 2.7 mmol/l (Table 2). Given this finding, the elevated serum potassium level was attributed to pseudohyperkalemia. Given that the white blood cell (WBC) count was lower than levels reported previously,[1] and the lack of evidence for other typical causes, the type of transport as an unusual cause was investigated. The next morning two serum levels were obtained simultaneously, one as sent via pneumatic tube transport and the other as walked down to the laboratory. One hour before, a whole-blood potassium level from a radial artery blood gas was obtained and sent through pneumatic tubing (Table 2).

Pseudohyperkalemia was initially noted by Hartmann and Mellinkoff.[2] They described several cases of patients with thrombosis with falsely elevated potassium levels. This phenomenon is defined as a discrepancy between the serum and plasma potassium of more than 0.4 mEq/l. It has subsequently been documented in other cases of thrombocytosis and leukocytosis.

Given the consequences of inappropriately treating pseudohyperkalemia, it is important to consider all possible causes of hyperkalemia. The cause of pseudohyperkalemia in cases of leukocytosis has been attributed to the fragility of the leukemic cells.[3] Pseudohyperkalemia attributed to pneumatic tube transport has been described in a patient with a WBC of 290K/μl.[4] The potassium level range was 6.2–9.1. In our case the WBC ranged from 136 to 184K/μl. We posit that the cause of the elevated potassium is the rupturing of fragile leukemic cells in pneumatic transport. Of note, in the previous case the patient was placed on dialysis with a potassium-free bath. At 45 min into treatment, the true potassium level was noted to be 3.2 mg/dl. The bath was changed to a 3K bath, however, the potassium level nadir was 2.3 mg/dl. There were no complications noted from the hypokalemia; however, this case shows the potential danger of initiating hemodialysis in a case of pseudohyperkalemia.

This case highlights the importance of considering all mechanisms, including transportation, of disruption of leukemic cells in cases of pseudohyperkalemia.

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